# PHILADELPHIA NEUROLOGICAL SOCIETY.

October 28, 1902.

The President, Dr. John K. Mitchell, in the chair.

*Astasia-Abasia.*—Dr. F. X. Dercum referred to a case which he had intended to present to the Society, but the patient had recovered. He was a colored boy, thirteen years old, and had been struck on the back with a ball. This was followed in the course of a month by great incoördination in the legs, and for seven months he was incapacitated for walking. He could move the legs well while in bed on his back. He made a perfect recovery under suggestion, without hypnotism, and the use of a placebo.

*Peculiar Swelling of the Back of the Hand.*—Dr. F. X. Dercum exhibited a woman who had a negative family history, was married, and the mother of two healthy children, and had had no miscarriages. Twelve years ago she had a prolonged illness of a doubtful nature. During convalescence she had swelling of both feet and hands. This swelling disappeared except in the right hand, where it still persists. The swelling has undergone no change. Her weight increased remarkably six months ago, and she gained one hundred pounds in the course of a year.

The dorsal aspect of the hand presents a soft gelatinoid swelling like that of myxedema. The finger nails are ridged and brittle, and somewhat atrophic. She has pain about the roots of the nails and the skin of the distal phalanges is somewhat shiny.

Dr. T. Diller reported a case of myasthenia gravis pseudoparalytica.

Dr. C. K. Mills reported a case with symptoms of myasthenia gravis, resulting from an electric shock of 1,300 volts.

Dr. S. W. Morton and Dr. C. K. Mills reported a case of acute bulbospinal paralysis.

Dr. T. H. Weisenburg read a paper on the effects of electricity on the human body.

*Bulbar Palsy.*—Dr. W. G. Spiller presented a man, aged thirty-two years, who in September, 1901, first noticed a sensation of fatigue in the muscles of the lower part of the face. He had been drinking previously. The sense of fatigue usually was not caused by eating or by cold, but by talking or reading aloud, and was worse in the morning. He complained also of tremor in the lips and neck on the left side, and fibrillary tremors in the left side of the lower part of the face had once been observed in the clinic. The man was unable to elevate either corner of the mouth, and when he laughed the corners of the mouth were drawn outward, but very slightly upward. Sensation on both sides of the face was normal. The reactions of the muscles of the face to the faradic current were normal. Dr. Spiller presented the case as one of bulbar palsy or myasthenia gravis pseudoparalytica in the early stages.

Dr. Wharton Sinkler remarked that the case reported by Dr. Diller was extremely typical, although the author had not laid stress on one of the most characteristic features of these cases, that is, the extreme exhaustion of the muscles after exertion. He spoke of the speech being worse after talking, but did not refer to the effect of muscular exercise. An interesting point in connection with the case was the occurrence of angioneurotic edema. This Dr. Sinkler considered favored the view advanced by some writers that myasthenia gravis is due to an infection.

With regard to the case reported by Dr. Mills, he said that it did not look to him like a case of myasthenia gravis, but more like one of true bulbar paralysis, from the fact that there was atrophy of the tongue, facial muscles, and of the upper extremity, with exaggerated knee-jerks.

Dr. D. J. McCarthy said that in the examination of the nervous system made in a case of myasthenia gravis of Dr. Burr, the findings were negative, but in that case and in another with autopsy since, distinct lesions in the thymus gland had been found. The first case developed after pregnancy, and there was a persistent thymus gland as large as the fist, and the lymphoid tissue throughout the body was increased in amount. Examination of the muscles gave negative results. In the second case, examination of the thymus showed enlargement with an abscess as large as a small walnut in the central part. Examination of the nervous system and of the muscles gave negative results. He referred to other reported cases in which similar lesions of the thymus gland had been found.

Dr. S. W. Morton said that the only reason for mentioning electricity as a probable cause in his case was that careful study had failed to reveal anything else as a cause. In the case of bulbo-spinal paralysis presented by Dr. Mills, the same cause suggested itself. It seemed possible that the frequent application of weak currents might exhaust the nervous system. Many of these cases of acute paralysis are undoubtedly due to auto-intoxication, or to some form of poisoning. With this idea in mind, he had looked for enlargement of the spleen, but found none. He mentioned that the pharyngeal reflex was impaired, but not lost.

Dr. Alfred Gordon read a paper on the condition of sensation in motor paralysis of cerebral origin, and reported the results obtained by a study of thirty-five cases.

Dr. F. X. Dercum remarked that the most common condition referred to by Dr. Gordon was hypesthesia. That hypesthesia is very frequently present in hemiplegia is a matter of common experience. This is especially true in old cases. A possible cause of the hypesthesia in these cases is deficient nutrition in the paralyzed limbs, which are cold and livid and the seat of arrested functional activity. Under such conditions we should look for lesion of the nerve endings and diminished conductivity of the nerve trunks.

Dr. Dercum did not think that anesthesia was necessarily in proportion to the motor loss.

Dr. Charles K. Mills said that while Dr. Gordon's paper was an interesting contribution to the question, it did not seem to him that, granting the accuracy of the observations, the conclusions apparently drawn were entirely warranted. Of the thirty-five cases examined, there were a number which failed to show any of the sensory disturbances referred to, and the author had made no attempt to explain the character of the lesion in those cases in which the symptoms were essentially motor. The speaker also called attention to the fact that no attempt had been made to separate the different varieties of astereognosis.

The majority of the cases of hemiplegia seen at the Philadelphia Hospital are due to subcortical lesions. Many are due to capsular, thalamo-capsular, lenticulo-capsular, and lesions of considerable extent, often hemorrhagic, which have acted in different directions, and there is every reason to believe that they involve the separate tracts which exist in the corona radiata, and in the capsule, and in other parts.

While not agreeing in the conclusions of the author, he held that this form of clinical work was of the greatest importance in the solution of these problems, especially when confirmed by autopsy.

Dr. F. Savary Pearce said that it had occurred to his mind that the anesthesia, the hypesthesia, and the other sensory changes occurring early in hemiplegia might be accounted for by the vasomotor paresis together with the formation of toxic products, and their retention in the tissue of the paralysed side, thus affecting the tactile corpuscles and the peripheral part of the nervous system. This part after a time partly or in whole recovers. In the chronic cases the sensory disturbances are probably the result of lack of nutrition, as suggested by Dr. Dercum, and not necessarily due to changes in the central nervous system. He believed that in many cases the lesion is in the peripheral parts rather than in the brain itself.

Dr. William G. Spiller said that the paper was purely clinical, and that we should not attempt to draw anatomical conclusions from it. In two of the cases in his own ward, which Dr. Gordon had employed in his study, Dr. Spiller obtained necropsies. In one there was an area of sclerosis in the occipital lobe, extending forward and involving the posterior limb of the internal capsule. In the other the optic thalamus was implicated.

In connection with Dr. Gordon's paper, Dr. Spiller referred to the recent paper by Marie and Guillain, the latter paper being anatomical. These authors had shown how difficult it is to determine the cause of hemianesthesia in hemiplegia, from the lesions found.

Dr. D. J. McCarthy referred to a case, the specimens from which he was at present studying. During life there had been a quadraplegia; no loss of sensation, although muscular sense was not studied. The autopsy showed hemorrhage affecting the lemniscus and a small area about it. The secondary degeneration extended upward, affecting the posterior part of the internal capsule. There was also some degeneration of the cortex, but this had not as yet been sufficiently studied, to permit an exact determination of the area.

Dr. Alfred Gordon said that it had not been his intention to solve the question of localization. He had wished simply to approach the subject from one side, and to study in an impartial manner the question whether in these cases of motor paralysis of cerebral origin, there were sensory disturbances or not. The literature shows such variations in the results that he wished to study the matter for himself. The question of separate localization is still far from being solved. As Marie has suggested, why should we not, as the evidence is so contradictory, cease fixing our minds on the *carrefour sensitif*, and look for sensory fibers in some other part; just as with the motor fibers, we remove a portion of the motor cortex and the patient recovers with complete return of power; this shows that the motor area is not the only portion of the brain which controls the motor nerves.

Dr. F. X. Dercum reported a case of adiposis dolorosa with involvement of the joints.